REMARKS ON EPILEPSY.

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IT is with considerable hesitancy that I approach this subject, fully comprehendiug its difficulties and my inability to adequately handle it.

The character of the times seems to render it almost impossible, in the great number of conflicting duties and occupations of the hour, for one to concentrate his time and mind on any one subject.

In the department of diseases of the nervous system, one at present finds it necessary to know more of general medicine outside of any special knowledge of this department itself than what a few years ago would have constituted a liberal education in medicine.

The object of my paper shall be to emphasize the idea of the cerebral character of epilepsy, and to place it in the class of organic diseases, rather than its generally accepted classification as a functional disorder.

As Gowers, in "Diseases of the Spinal Cord," well says: "Strictly speaking, a functional disease is one which consists in a disorder of function without any preceding alteration of nutrition," and affections of the nervous system which can be included under this head are very few.

In most cases of so-called functional disease we must assume changes in nutrition.

Ross, in the last edition of his work on nervous diseases, defines epilepsy as a chronic functional disease of the nervous system, characterized by loss of consciousness, and convulsions.

Krafft-Ebing, in his "Lehrbuch für Psychiatrie," describes it as a disease marked by repeated attacks of complete or incomplete loss of consciousness, or even slight dulness of intellect, accompanied by partial or general convulsions dependent on spasm of the cerebral arteries.

Brown-Séquard has, perhaps, given us the most satisfactory definition. He says: "Epilepsy seems to consist in an increased reflex excitability of certain parts of the cerebro-spinal axis, and in the loss of control that in the normal condition the will possesses over the reflex faculty."

He places its cause as in the medulla.

The disturbance of the functions of the cerebral lobes during and immediately after a fit, and in the interparoxysmal period, he ascribes as due to alteration in the cerebral substance taking place during the seizure.

The hitherto mysterious coincidence of loss of consciousness, or, in other words, loss of function of the cerebral lobes, with increased action of the medulla, in an epileptic attack, is thus easily explained.

In describing epilepsy, great stress is laid on the character of the seizure, the attending epileptic cry, the unilateral or bilateral form of the convulsion, and but little attention is paid to the previous mental state, or to the condition in the intervals between the attacks—that which is called by Nothnagel the epileptic change, or, still better, by Griesinger the epileptic condition.

I do not refer to the status epilepticus, that condition immediately following a paroxysm, in which the patient may be in a state of mania, or in which paroxysm may follow paroxysm for hours or days, but I mean the period intervening between the attacks, be it six days or as many years.

The question is, then, What produces this epileptic condition, and what the outbreak of the seizures?

It seems to me the confusing of these two has caused the confusion in our ideas of the nature and seat of the disease.

Unconsciousness and convulsions may be produced by various causes. We know excessive loss of blood, ligature of the carotids, local or general cerebral anæmia, may account for them.

Bernard, on cutting the sympathetic in the neck and extirpating the superior cervical ganglion, caused an increase of temperature on that side of the brain.

Nothnagel found that, after performing the above experiment, irritation of the crural nerve caused contraction of the vessels of the pia mater, and while cutting of the sympathetic caused the dilatation of the vessels, faradization of the cut extremity caused narrowing. Van der Beke and Callenfels obtained similar results. The author then states:—these experiments prove three things: first, that the vasomotor nerves for the vessels of the pia course in part through the fibres of the sympathetic in the neck; and, second, that perhaps another set, more important, pass through the superior cervical ganglion; and, lastly, that above the ganglion are fibres probably accompanying the cranial nerves.

This would explain, in the reflex epilepsy following teething, intestinal irritation, and injuries, the contraction of the vessels of the brain which follows.

There may be other causes, however, for convulsions besides cerebral anæmia. The cerebrum can be removed, and yet convulsions follow. Marshall Hall long ago stated that anæmia of the medulla oblongata was the cause of convulsions. There is no doubt that irritation of the medulla can cause them, and were this all of epilepsy, we might well rest our case with this part of the nervous system as the seat of the disease. Nothnagel has proven the existence of the convulsive centre in the medulla, and the presence also of the vasomotor centre explains, by its producing cerebral anæmia, the accompanying unconsciousness, but this does not explain the intellectual deterioration present, the epileptic condition to which I have already referred, and which I regard as the most essential feature of the disease.

Cerebral congestion was for a long time held as the cause of epilepsy, but Brown-Séquard, Bernard, Schiff, and others have proved that this is secondary to the convulsion.

Trousseau says of this:—there are therefore two very distinct conditions in an attack of eclampsia or epilepsy,

whether idiopathic or symptomatic: first, a cerebo-spinal modification unknown in its essence and its nature, which in a second abolishes all the manifestations of animal life; and, second, a secondary cerebral congestion, which, although less important, may in some extremely rare cases be carried so far as to produce subcutaneous ecchymoses, cerebral capillary hemorrhage, and even meningeal hemorrhage. In the interval between the seizures there is a change in the individual. We may have mania or delirium continuing for weeks; there is a marked loss of intellectual power, and of memory, an indefiniteness of ideas, an irritability of temper, or, as Esquirol puts it, if not insane, the character is peculiar, irritable, capricious, paradoxical, the features become coarse, a precocious senility sets in.

Who has not observed the obstinacy of the epileptic, the look of vacancy or fatuity, the mental weakness?

In the cases that have come under my observation in the last five years, few if any have preserved their full intellectual power. This would speak against the theory of regarding the seat of the disease as in the medulla.

Extensive implication of the medulla, as in primary and secondary bulbar paralysis, does not involve the intelligence. I have two cases of that nature at present under observation, where the intelligence is absolutely intact.

I also protest against calling a single convulsive seizure caused by teething or indigestion, as epileptic.

Should these attacks continue, then, as a result of repeated sensory irritation conveyed to the brain, the cerebral cortex may become involved. Chronicity must be present in this disease as a factor.

The cortex is the location of all psychical processes which are present to our consciousness; it is the seat of memory and all acts of the will (Edinger).

In general paralysis Tuczeck shows the first layer of the cortex is first involved, and so in order to the fourth. Not until the first month of life, however, do the cerebral fibres assume a sheath, and until then the child's acts are all reflex.

The fibræ propriæ or association fibres are the first to as-

sume this sheath, and these fibres play an important part in the extension of an epileptic seizure.

When we have simply loss of certain mental functions, as paroxysmal attacks of loss of words, or loss of the power of reading, which may precede the seizure or be present without the latter, we may well conceive that certain convolutions containing the centres of these attributes are affected.

Should this extend its irritative action, the surrounding centres controlling motor or sensor functions might be involved, and thus lead to a more extended class of symptoms.

Epilepsy *per se* is a chronic condition or disease of the cerebral cortex, manifesting itself by periodical attacks of convulsions, etc., with a progressive intellectual decline and tendency towards insanity, and belongs to the class of cases coming under the head of general paresis.

Simple convulsions are distinguished by their constant relation to some known cause, and by their yielding to treatment directed against it. Hitzig speaks of the continuance of the epilepsy after the removal of the irritation. I think this sustains the idea of a morbid process having been established. The cause may have been removed; the condition, however, has been induced, and now we find the permanent symptoms of the disease present.

The tendency at present is to regard the loss of consciousness, be it slight or great, with or without spasm, even dizziness,—in fact, any alteration of mental activity occurring paroxysmally, as epilepsy. I certainly hold with this view. I would look upon the epileptic seizures as simply expressions of the disease as existing in the degeneration of the gray matter of the cortex. It may involve any part, as the centre of speech or a motor centre, causing loss of control over the lower centres in the medulla, and thus lead to convulsions. Just as Bright's disease is accompanied by uræmic convulsions, the latter not being the disease but simply an expression of the diseased condition of the kidney, which may produce a large number of other changes in the system, so in epilepsy, we have various

consequences of the morbid state express themselves in convulsions, mental weakness, or even insanity. In the one we have arrived at a definite knowledge of the pathological changes, in the latter we have not as yet, but should look for them in the cortex. If in the frog, after removal of the cerebrum, the reflex acts are increased, it is fair to presume that in epilepsy, the cerebral centres being for the time in abeyance, the lower spinal and medulla centres, being no longer inhibited, are subject to increased reflex action on the slightest external irritation. Let the cerebral loss of function be produced by anæmia, or otherwise, does not affect the principle. If we consider that cerebral irritation is capable, independent of lesion of the medulla, of producing convulsions and loss of consciousness, it seems more probable, in consideration of the many mental symptoms present in epilepsy, and which can only have their origin in the cortex, that the cerebrum is also the original seat of the convulsions.

Hysterical attacks, although often as violent as epileptic convulsion, do not lead to any condition similar to that of epilepsy.

Who has not observed the tonic and clonic contractions in hysteria, involving the face, extremities, and trunk, and even partial loss of consciousness? and yet, although this may be often repeated, the after-effects never resemble those seen in epilepsy. The definition of the disease as given in the earlier part of this paper, in which it was described as a sudden but temporary loss of function of any or all the cerebral centres, is hardly correct, as implying that between these attacks a condition of restoration of cerebral function exists. As Krafft-Ebing has well put it, the epileptic shows a psychical degeneration, a loss of intellectual power up to complete imbecility, showing itself by forgetfulness and slowness of judgment and perception. I would, therefore, define epilepsy as a disease of the cerebral cortex, attended by a progressive decline of the intellectual powers and by paroxysmal attacks of partial or complete loss of consciousness, accompanied by convulsions involving a part or the whole of the body.

In reference to the treatment of epilepsy I depend on the bromides exclusively, usually commencing with thirty-grain doses three times a day.

I prefer, as a rule, the mixed bromides, in the proportion of four parts of the potassium bromide to two parts each of the sodium and ammonium bromide; when cardiac weakness is present, employing digitalis or aromatic spirits of ammonia.

I have not found, as some affirm, that iron acts deleteriously—that is, when anæmia is present. Nor does the continued use of the bromides for years even, in my experience, lead to any mental disturbance; in fact, I find a gradual improvement in this respect whenever I succeed in controlling the disease.

In regard to cure of the disease, in my opinion that is less often accomplished than amelioration of the condition.